

Interdependence **of Blood Pressure, Weight Gain, and Fetal Weight** *During Normal Human Pregnancy*

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THE CLINICALLY significant changes in blood pressure and weight gain during pregnancies that are uncomplicated by toxemia remain in debate. Since 1899, when Weisner's observations on blood pressure in pregnancy were cited (1), various changes have been reported. Our present teachings, as indicated by current textbooks, vary from a minimal decline in the diastolic pressure in the midtrimester (2-4) to a decrease of 10-15 mm. of mercury (Hg) in systolic and 5-15 mm. Hg in diastolic blood pressures (5-6), with a return to prepregnancy or first trimester levels at term. Hytten and Leitch (7) described a small decrease in systolic blood pressure early in pregnancy, rising late in preg-

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nancy, with the diastolic pressure being "considerably" below nonpregnant levels from early pregnancy until the last few months, when it rises toward nonpregnant levels. Thus an obstetrician, depending on what he reads, may interpret a significant rise in blood pressure during the third trimester as either a physiological change, a return from "the midtrimester drop," or a pathological change.

Recent correlations between maternal weight gains and fetal weight (8, 9) have emphasized a need for clarification of the physiological weight gain in pregnancy and its relationship to fetal weight. Chesley (10) noted the mean net gain of 24 pounds, with a 2-pound gain in weight during the first trimester, followed by 11-pound gains in both the second and third trimesters. Thomson and Billewicz (11) reported similar findings in normotensive primigravidas, with a mean gain of 24 pounds from the 13th week of gestation to term.

Other investigators have reported a loss of 2 pounds or more in the 2 to 3 weeks before the onset of labor (12), and an inconsistent initial loss during the first trimester (3a). The increased incidence of toxemia associated with either excessive or minimal gains in weight has been well demonstrated (11, 13). Total gains in maternal weight correlate directly with mean fetal weights at term (3a, 9). The stage of pregnancy during which weight gain influences the fetal weight at term remains unclear.

Because of these conflicting reports, we undertook this study to answer the following questions:

1. What is the blood pressure as measured by the sphygmomanometer throughout normal (non-toxemic) pregnancy?
2. Is the blood pressure throughout pregnancy different in patients with excessive or minimal gains in weight?
3. What is the physiological gain in maternal weight throughout pregnancy, and does the pattern vary in patients with excessive gain compared with those having a minimal gain in weight?
4. At which stage of pregnancy does maternal weight gain correlate most closely with term fetal weight?

Materials and Methods

A total of 344 patients was observed during 544 pregnancies; 181 patients were nulliparous and 163 were multiparous. A registered nurse measured blood pressures of patients in a seated

position with a standard mercury sphygmomanometer and stethoscope after the patients had been seated in the waiting room for variable lengths of time. The patients, fully clothed except for shoes, were weighed on a balance scale. All prepregnancy weights that were analyzed were measured in the same manner during a gynecologic appointment within 1 year of the study pregnancy.

A total gain in maternal weight of more than 30 pounds was arbitrarily designated as excessive, while a total gain of less than 11 pounds was designated as minimal. No patient developed preeclampsia, and all patients were managed without diuretics, antihypertension agents, or appetite depressants. Fetuses were weighed on a balance scale immediately after birth. No restriction in weight or diet was imposed on any patient. Patients with known prepregnancy hypertension, heart, renal, or metabolic diseases were eliminated from analysis. Hendricks (author) examined and delivered all patients at MacDonald House in the University Hospitals of Cleveland, Ohio. No patient was excluded from analysis for excessive gain in weight or development of hypertension during the study pregnancy.

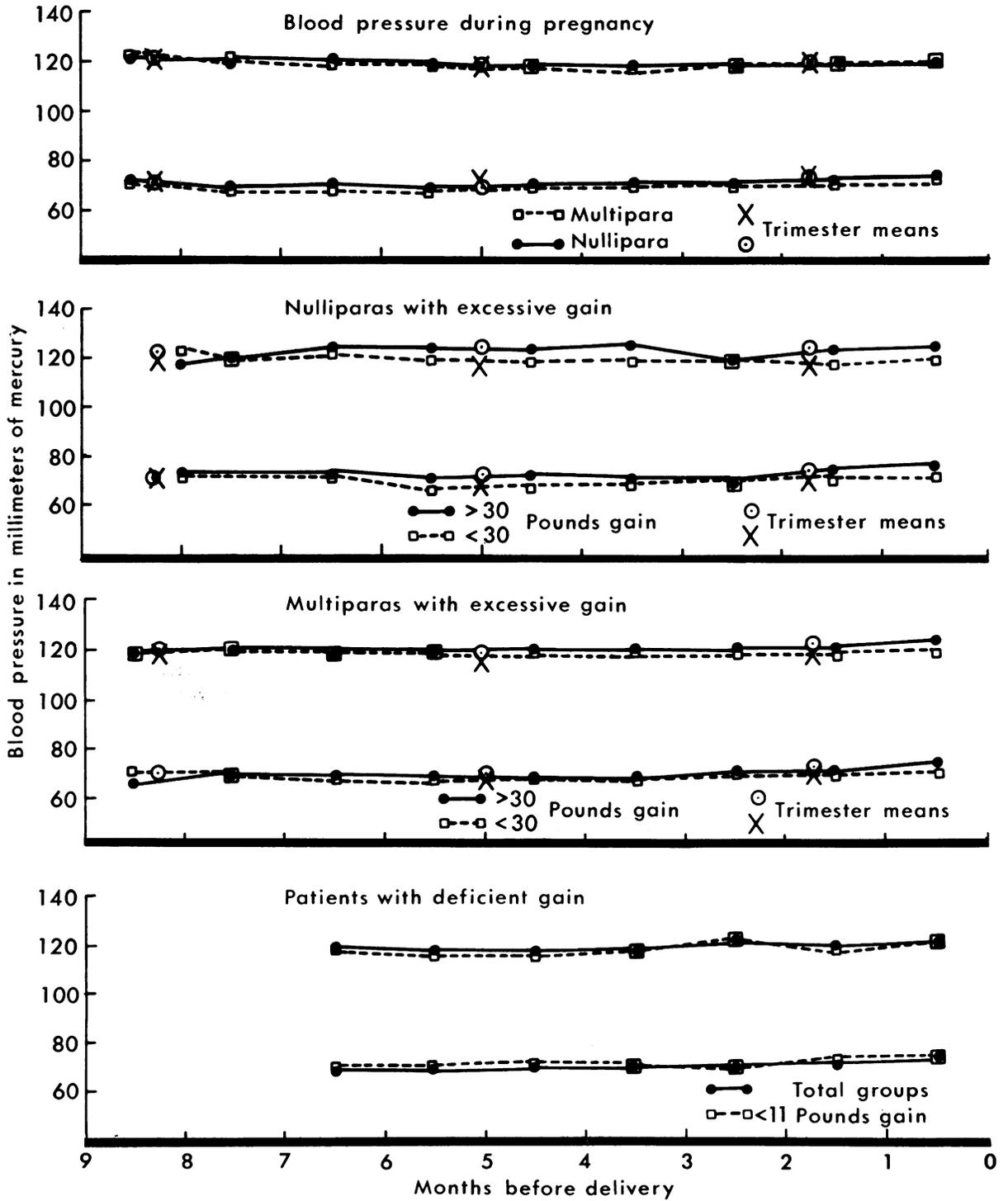
Results

Blood pressure. The mean systolic and diastolic blood pressures for nulliparas and multiparas, by months before delivery, are shown in figure 1. No statistically significant differences or changes in these blood pressures were observed throughout pregnancy. Also, the mean systolic and diastolic blood pressures during the trimesters (see table) were not significantly different. No outstanding trends were noted when reviewing the individual records of the patients.

Mean systolic and diastolic blood pressures measured by sphygmomanometer, by parity, in each trimester for 544 pregnancies without toxemia, mm. Hg

Trimester	Nulliparas	Multiparas
First:		
Systolic.....	121.7	120.5
Diastolic.....	70.3	69.9
Second:		
Systolic.....	118.3	117.5
Diastolic.....	68.6	67.8
Third:		
Systolic.....	118.4	118.5
Diastolic.....	71.6	69.9

Figure 1. Influence of total weight gain on blood pressure throughout pregnancy



Effect of weight gain on blood pressure. Patients with minimal or excessive gains in weight did not have mean systolic and diastolic blood pressures significantly different from those of the total group. Blood pressure means in nulliparas and multiparas (fig. 1) gaining more than 30 pounds, compared with those gaining less than 30 pounds, throughout pregnancy were not significantly different. Patients with minimal gains in weight had blood pressures insignificantly different from those of patients who gained more than 11 pounds (fig. 1).

Weight gain. The cumulative weight gain throughout pregnancy was determined (fig. 2). A small gain occurred in the first trimester, followed by an increasing rate in the second trimester and a constant rate in the third trimester. The mean gain in weight for these patients was 22.7 pounds at the time of delivery.

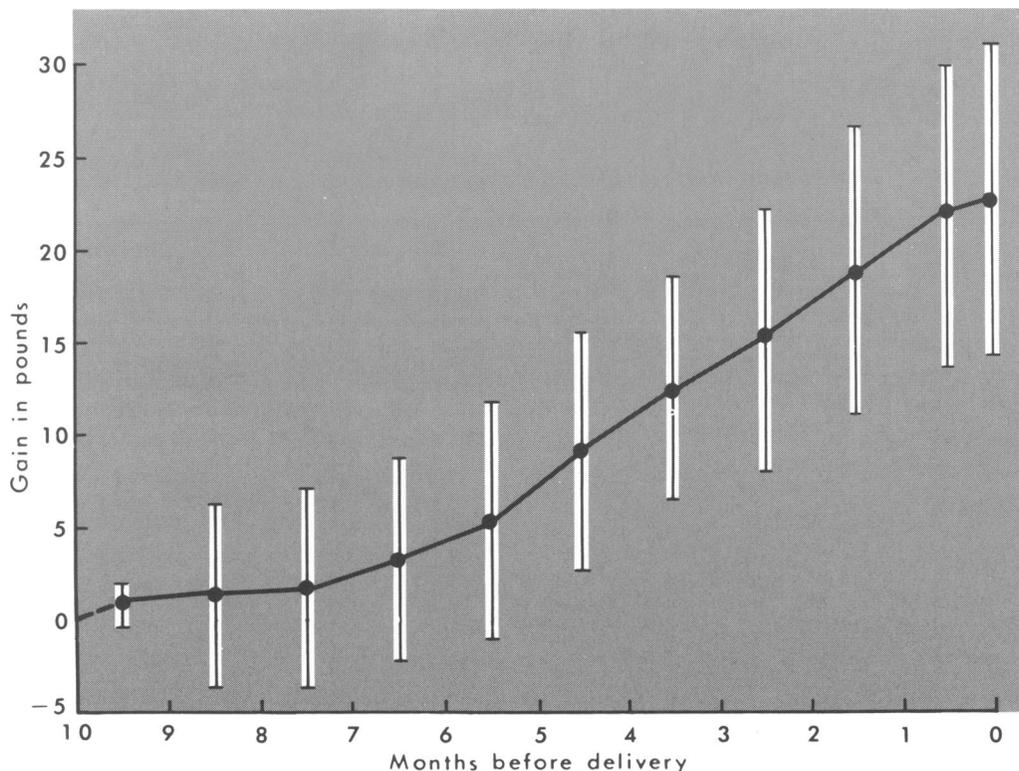
Nulliparas gained slightly more weight than multiparas in all three trimesters, but the pattern of gain throughout pregnancy was similar (fig. 3). The mean prepregnancy weight was 125.47 pounds for nulliparas and 128.55 pounds for multiparas, with a mean total gain of 24.03 pounds

for nulliparas compared with 22.02 pounds for multiparas.

The pattern of cumulative weight gain for those women having excessive gains was different from that of the whole group and of those with minimal gains (fig. 4). Patients with excessive gains had a prepregnancy mean weight of 124.9 pounds for nulliparas and 126.7 pounds for multiparas. The pattern for those who gained excessively, compared with that for the total group, was an increased rate of gain that started in the first trimester and continued throughout pregnancy. Only 4.5 percent of the patients with excessive total gain in weight failed to show an increased rate of gain in the first trimester. No differences in patterns of excessive gain throughout pregnancy could be demonstrated between nulliparas and multiparas (fig. 5).

The curve showing weight gain of patients with minimal total gain (less than 11 pounds) was different from that of the whole group. The mean prepregnancy weight of these patients was 142.3 pounds for nulliparas and 140.9 pounds for multiparas. Compared with that of the total group (fig. 4) the pattern of these minimal gainers of weight

Figure 2. Mean cumulative weight gain (\pm S.D.) throughout 544 pregnancies without toxemia



was a loss in the first trimester and a slower rate of gain in the second and third trimesters.

Effect of maternal weight gain on term fetal weight. Total gain in maternal weight was correlated with term fetal weight (fig. 4). Patients gaining less than 11 pounds had a mean term fetal weight of 3,082 grams, compared with 3,791 grams for patients gaining more than 30 pounds and 3,398 grams for the whole group.

To determine the effect of loss in maternal weight during the first trimester on term fetal weight, the patients who had gained or lost weight in the first trimester were grouped separately (fig. 6). These two groups were subdivided, depending on whether the total gain was greater than 30 pounds or less than 11 pounds. The mean term fetal weights appeared to relate to maternal gain in the first trimester, total gain, and gains in the second and third trimesters. Loss of maternal weight in the first trimester and minimal total gain were associated with the smallest infants, while weight gain in the first trimester followed by excessive total gain was associated with the largest infants.

Patients who lost weight in the first trimester and gained more than 11 pounds during the second and third trimesters did not deliver infants with a mean weight different from that of infants

born to patients who gained weight in the first trimester but failed to gain a total of 11 pounds. No patient gained excessively after an initial loss of weight in the first trimester.

Discussion

Blood pressure. No statistically significant variations in blood pressure were noted throughout pregnancy. Bordley and associates (14) have emphasized the limitations of the sphygmomanometer as ± 8 mm. Hg when compared with direct intra-arterial measurement.

Using intra-arterial catheters, Burwell and associates (15) noted a decrease of 5–14 mm. Hg in the diastolic blood pressure during the middle and early third trimesters. They reported that the systolic blood pressure remained constant with a resultant increase in pulse pressure.

Studies in which sphygmomanometers were used had variable results. Landt and Benjamin (16), in their study of 24 patients, noted a mean decrease of 10–15 mm. Hg in systolic and diastolic blood pressures in mid-pregnancy. In a population of private patients similar to the one in this study, Andros (17) noted a persistent decrease of 4 mm. Hg in mean diastolic blood pressure from prepregnant levels during the first and second trimesters, but he eliminated all patients

Figure 3. Mean cumulative weight gain of nulliparas and multiparas, by parity, throughout 544 pregnancies

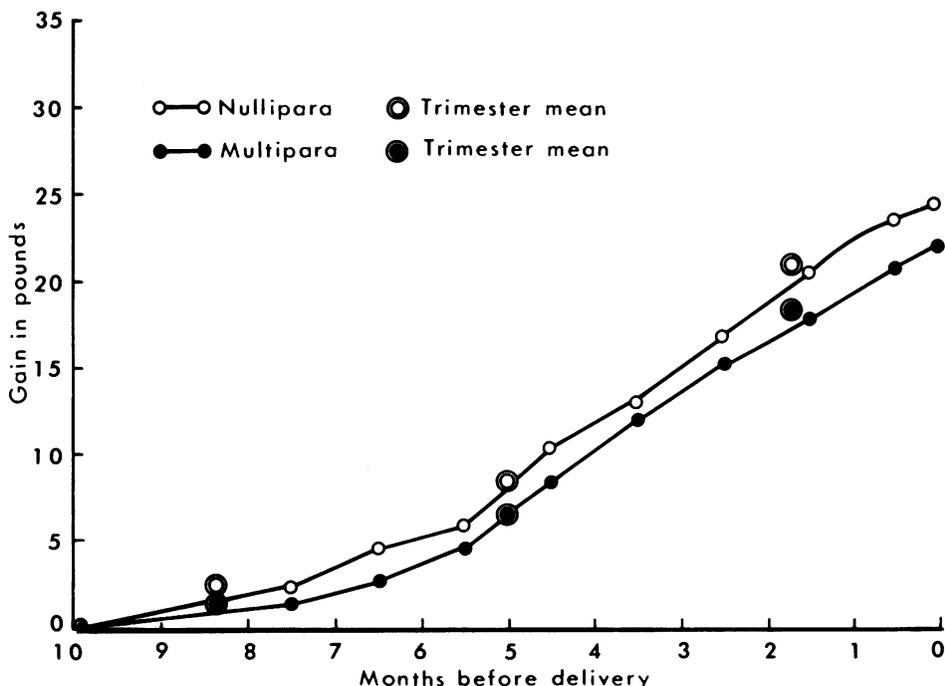
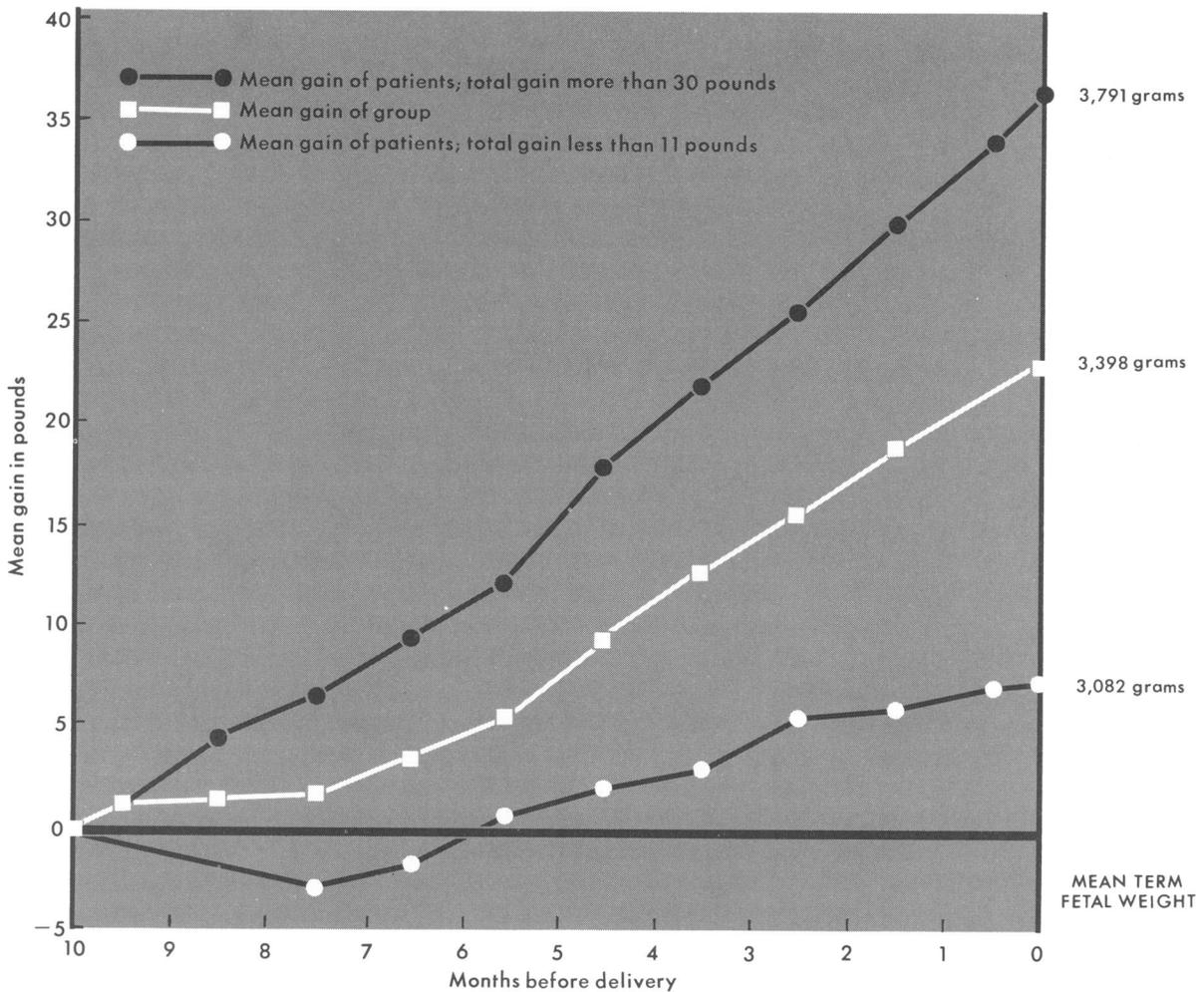


Figure 4. Patterns of excessive (more than 30 pounds) and deficient (less than 11 pounds) weight gains throughout 544 pregnancies, and mean fetal weight



with blood pressures greater than 140 mm. Hg systolic and 90 mm. Hg diastolic. No significant change in blood pressure was noted by Henry (18). He and Slemons and Goldsborough (19) were also unable to demonstrate a difference between the blood pressures of multiparas and nulliparas.

Although we noted small differences in blood pressure means between the second and third trimesters, these differences were not only statistically insignificant but were well within the range of error of the sphygmomanometer. More importantly, however, it is not within the physician's ability to interpret a change of this magnitude as a change in the patient's physiological status, nor can it be confused with an impending pathological state.

Other investigators have correlated an excessive total gain in weight, an increased gain in the first

trimester, and a minimal gain in total weight with elevated maternal blood pressure. Thomson and Billewicz (11) have demonstrated that patients with preeclampsia and other hypertensive disorders have a continuous excessive rate of gain throughout pregnancy. Vedra (20) also reported an increase in first trimester weight gain in patients who developed toxemia later in pregnancy. Tompkins and co-workers (13) have shown the correlation between both excessive and deficient gains in maternal weight and an increased incidence of toxemia.

No patients developed toxemia, and no statistical differences were noted in blood pressures throughout the pregnancy of patients with a total gain in weight greater than 30 pounds or less than 11 pounds. Patients with a gain of less than 11 pounds were significantly heavier before pregnancy than the total group. Because no

difference in blood pressure could be demonstrated in this group, we concluded that the excess initial weight did not influence blood pressure nor cause significant artifacts with the sphygmomanometer. None of the patients with a low gain in weight, compared with patients in the series by Tompkins and co-workers (13), was underweight at pre-pregnancy examination. Therefore, in this well-nourished group of private patients, even widely varying patterns of gain were unrelated to significant changes in blood pressure.

Weight gain. The total gain in maternal weight has varied in different populations. Chesley (10) reported a mean gain of 24 pounds, while Eastman and Jackson (9), in a large series, noted a mean gain of 21.8 pounds in white patients as compared with 19.9 pounds in black patients. In our study of white patients, none of whom had toxemia, the mean gain was 22.02 pounds for multiparas and 24.03 pounds for nulliparas.

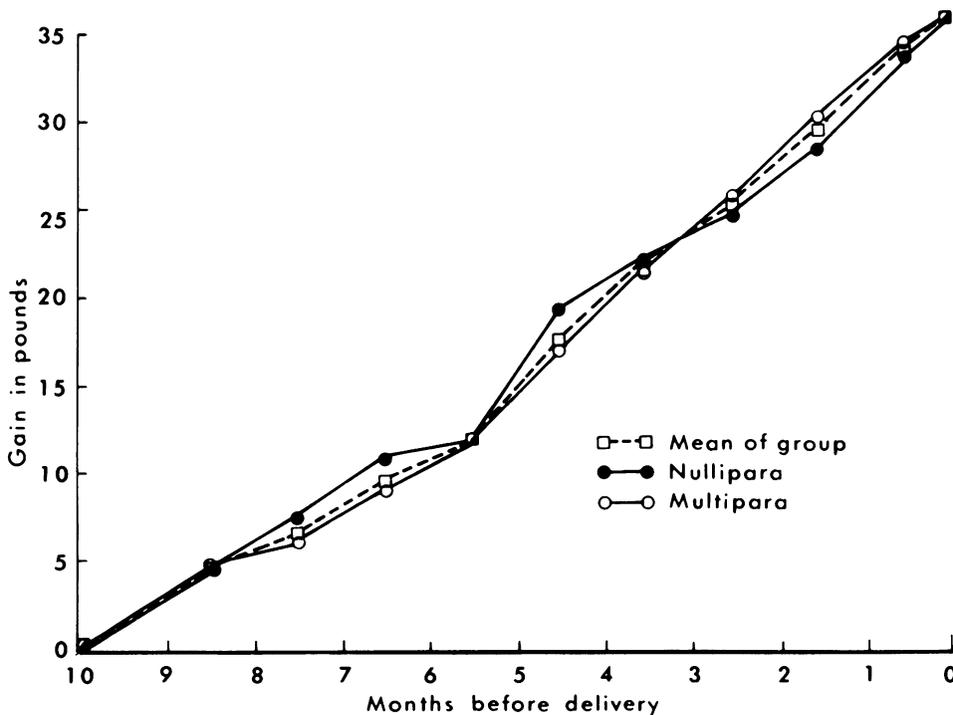
The rate of weight gain for our patients was slightly less and the pattern of gain in the first 12 weeks was different from those of patients in the series of Thomson and Billewicz (11) and Tompkins and associates (13). The decreased rate of gain in the last 2 weeks of pregnancy, noted by Tompkins and associates (13), was not observed

in this study. In the first 10 weeks of gestation there was minimal gain in weight, followed by an increasing rate of gain during the 10th to 18th weeks of gestation, followed by a constant rate of gain until delivery. Our patients gained about 0.7 pound by the 6th week of gestation compared with the smaller gain in weight found by both Thomson and Billewicz (11) and Tompkins and associates (13) when they extrapolated the mean weight at 12 weeks of gestation to zero at the time of the last normal menstrual period.

The differences in total weight gain and in patterns of weight gain between nulliparas and multiparas were not statistically significant. O'Sullivan and associates (21) were also unable to demonstrate any significant difference in total gain by parity when a correction for pre-pregnancy weight was used.

The known components of the weight gain are best demonstrated (fig. 7) by using results of many investigators, showing the portions of weight gain attributable to fetal weight (22, 23), placental weight (7a, 22), amniotic fluid (24, 25), breasts (7b), uterus (26), and blood (27). The blank area includes adipose tissue and intracellular and extracellular water in other organ systems. This presentation is somewhat deceptive in that it

Figure 5. Mean cumulative weight gains of nulliparas and multiparas gaining more than 30 pounds throughout pregnancy



gives the impression that all the components are increasing at about the same rate and that they make up the same proportion of weight gain throughout pregnancy. Figure 8 was constructed from the same data (7a, 7b, 22-27) to demonstrate the percentages of total gain in weight attributable to these components at any given time in gestation. Most of the early gain comes from increased uterine weight, generalized intracellular and extracellular fluid, breasts, and amniotic fluid. The placenta and the "payload of pregnancy," the fetus, account for little of this early gain. This pattern continues until about the 20th week, when the situation is reversed. During the time of maximum gain in maternal weight, the supporting systems begin to account for a progressively smaller proportion of the total gain in weight while the fetus accounts for an increasing proportion.

Effect of maternal weight gain on term fetal weight. The observations of Eastman and Jackson (9) concerning the correlation between total ma-

ternal weight gain and fetal weight indicate the possibility of a cause-and-effect relationship. The higher incidence of neurological damage in low birth weight infants is of obvious concern. Patients who gain weight poorly have a higher incidence of low birth weight children as well as children of lower mean birth weight. It remains to be demonstrated, however, that by having a patient gain more weight than she ordinarily would the birth weight can be increased and neurological problems decreased. One must first be able to identify the patient whose natural course is a minimal gain in weight during pregnancy.

The pattern of cumulative weight gain for patients who gained a total of less than 11 pounds is shown in figure 4. Characteristically, they lost weight in the first trimester and gained a little by midpregnancy, when they were gaining weight at their maximum rate. Children born to mothers with this pattern of minimal weight gain were significantly smaller than those of the whole group.

Figure 6. Mean term fetal weight, with loss or gain of maternal weight throughout pregnancy

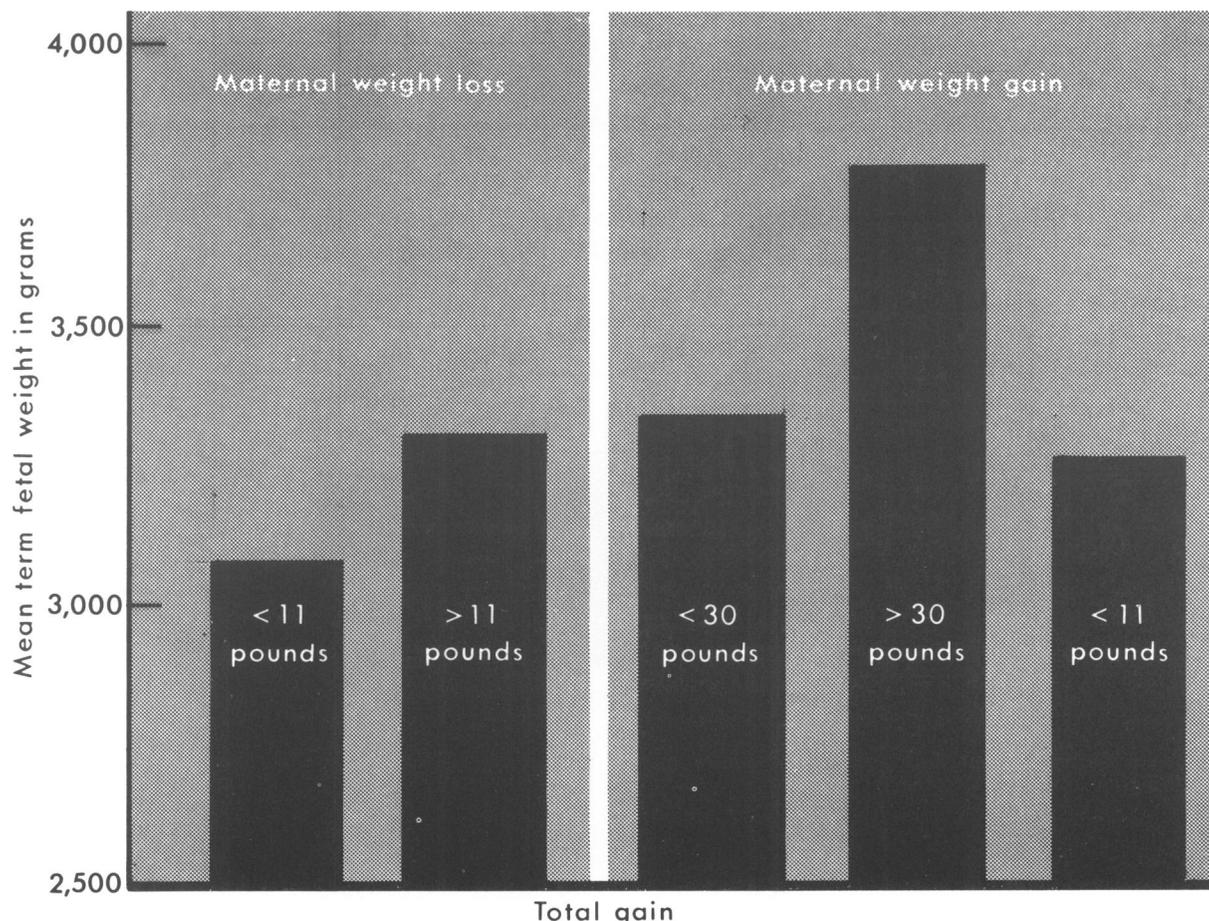


Figure 7. Mean weight of components of maternal weight gain throughout pregnancy, as measured by other investigators (see text)

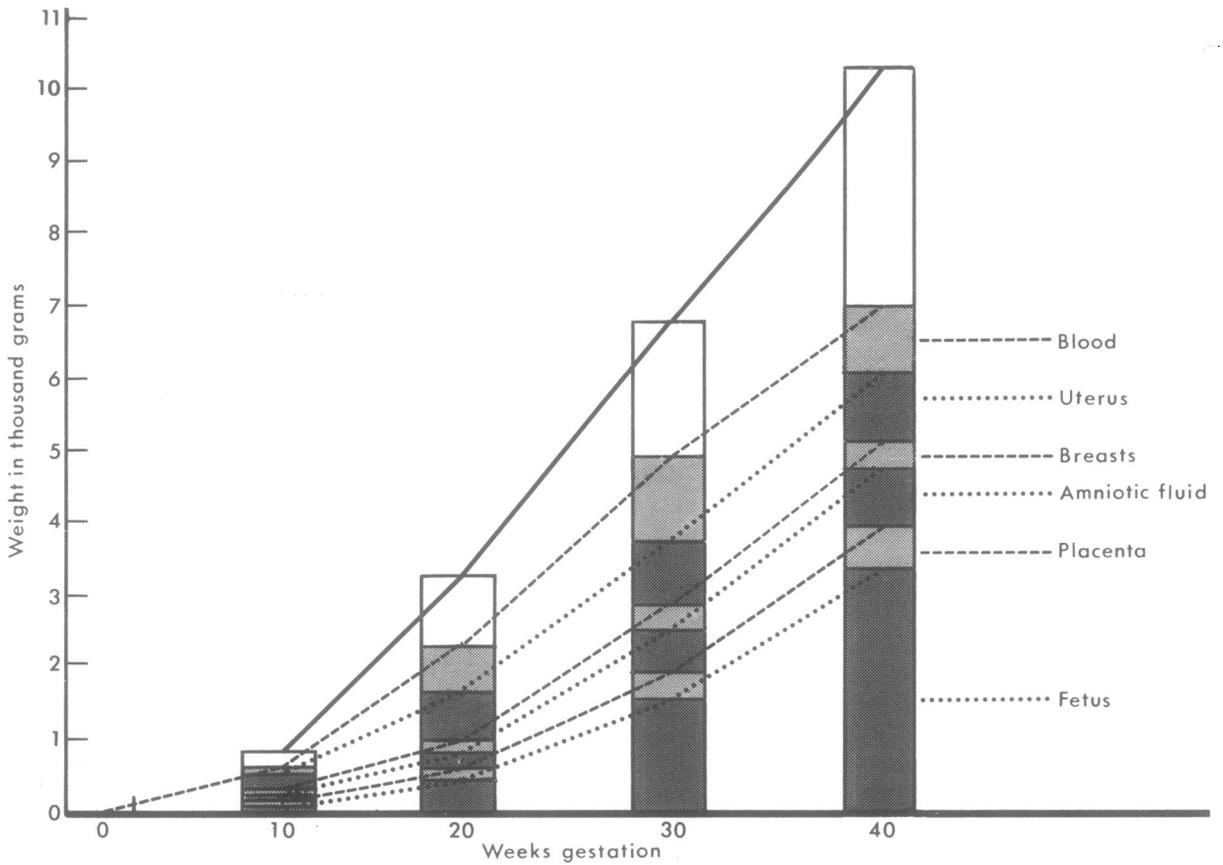
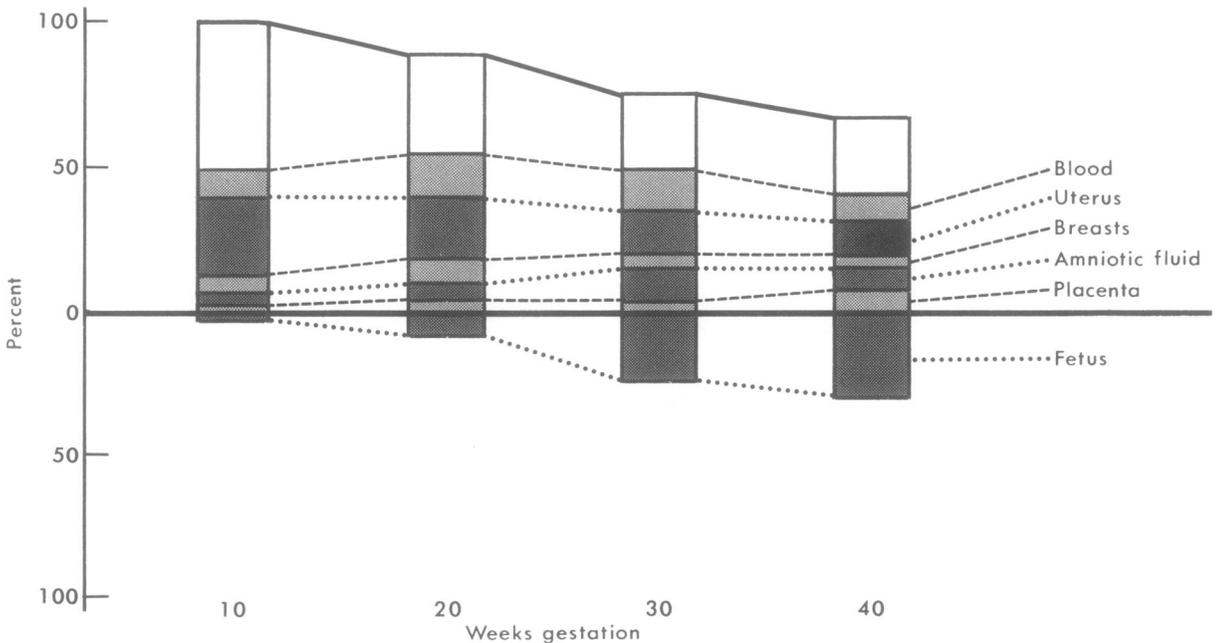


Figure 8. Percentage of total weight gain at various stages of pregnancy attributed to individual components of weight gain, as measured by other investigators (see text)



Women gaining more than 30 pounds had a different pattern of weight gain and significantly larger children. A steady, rapid rate of gaining weight, beginning in the first trimester, continued to term. It appears that a large percentage of patients who are going to gain minimal or excessive amounts of weight can be recognized in the first trimester, and attempts could be made to alter the total gain.

Mean prepregnancy weights were significantly less for the 125-pound patients who gained excessively as compared with the 141-pound patients with a minimal gain. Although no diets were advised, some patients may have unconsciously changed their diets or failed to report a change. Many obstetricians would have considered our minimal-gain patients overweight and would have placed them on weight-reducing diets. This practice must be questioned, however, because even though these patients were heavier, the fact that they gained less than 11 pounds throughout pregnancy resulted in a significantly smaller child.

Term fetal weights appeared to be related to the amount of weight gained at various phases of pregnancy as well as to the total weight gain. Patients with consistently high rates of gain throughout pregnancy had the largest children. Loss of weight in the first trimester, and a low rate of gain throughout the remainder of pregnancy was associated with the lowest mean term birth weight. Loss of weight in the first trimester, low weight gains in the second and third trimesters, or moderate gains in weight throughout pregnancy were associated with fetuses of intermediate weights. Gaining less than maximal weight at any time during pregnancy was associated with an infant of less than maximal weight.

If weight loss in the first trimester, characteristic of deficient weight gainers, changed to a normal weight gain in the last two trimesters, the mean fetal weight was essentially the same as for fetuses of patients with a gain in the first trimester and a moderate total gain. These results indicate that if one were able to obtain a moderate gain in maternal weight in the second and third trimesters in a woman with a loss of weight in the first trimester, the mean fetal weight would be increased. The supporting systems developing in early pregnancy may not be so dependent on gain in maternal weight that the mean fetal weight at term cannot be influenced in later pregnancy.

A patient gaining normally in the first trimester, with minimal gain in total weight, has a mean

term fetal weight that is not significantly different from that of a patient with a moderate gain in weight in all trimesters. This observation indicates that if limitation of weight were advisable in a patient gaining in the first trimester, reducing the weight gain throughout the remainder of pregnancy would result in an intermediate-sized infant. These patients spontaneously changed their weight-gaining patterns, however, and whether a patient with a deficient gain in weight in the first trimester can, through education, better nutrition, and encouragement, gain more weight, increase the weight of the fetus, and decrease the chance of prematurity and neurological damage remains to be demonstrated.

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A total of 544 pregnancies in 344 well-nourished private patients without toxemia was followed, using a balance scale and a sphygmomanometer, to determine (a) the blood pressure throughout pregnancy, (b) the effects of excessive (more than 30 pounds) and minimal (less than 11 pounds) total gain in maternal weight on blood pressure throughout pregnancy, (c) the physiological pattern of maternal weight gain, and (d) at

which stage of pregnancy maternal weight gain correlates most closely with term fetal weight.

The mean systolic and diastolic blood pressures did not vary significantly throughout pregnancy, and no effect of parity or weight gain was noted. The physiological pattern of weight gain was a minimal gain in the first trimester, followed by a rise to a constant rate in the second trimester, and a continuation of this rate in

the third trimester until delivery. Patients gaining an excessive or minimal amount of weight had different patterns of weight gain throughout pregnancy that could be differentiated before 20 weeks of gestation. Although mothers with a pattern of excessive weight gain had the largest children, a decreased rate of gain at any stage of pregnancy was associated with a lower mean fetal weight.